

## HERPES SIMPLEX VIRUS

### I. INTRODUCTION

Herpes genitalis is a sexually transmitted disease caused by the herpes simplex virus (HSV), a DNA virus that has two serotypes: HSV-1 and HSV-2. HSV-1 is responsible for virtually all cases of oral herpes and for approximately 50% of the first episode of genital infections. HSV-2 is the principle serotype that causes recurrent or subclinical genital infection. Genital herpes infections may be classified as primary, nonprimary, and recurrent.

- A. Primary infection represents the individual's first exposure to HSV and is characterized by constitutional symptoms and multiple painful vesicles on the vulva, vagina, and/or cervix. Lesions may occur between 2 and 14 days following exposure to infectious virus. These lesions tend to resolve within 3 weeks. Shedding of the virus from the lower genital tract of women occurs during the first 3 months after primary genital lesions have healed, although subclinical (asymptomatic) viral shedding can continue indefinitely.
- B. A nonprimary herpes episode is a HSV infection that does not behave clinically like a symptomatic primary infection. There are fewer systemic manifestations, less pain, a briefer duration of viral shedding and a more rapid resolution than primary herpes. These episodes may be the result of an initial HSV-2 infection in the presence of partially protective HSV-1 antibodies.
- C. Recurrent herpes infections typically produce minimal constitutional symptoms, fewer lesions, and more rapid resolution. Recurrent herpes is caused by reactivation of latent viral infection and is manifested by a characteristic prodrome followed by a limited vesicular eruption. Shedding of the virus from the genital tract without symptoms or signs of clinical lesions (subclinical shedding) is episodic and last on the average of 1.5 days. Subclinical shedding makes this viral STD difficult to control and prevent.

Neonatal HSV infection is one of the most significant sequelae of HSV infection in an adult woman. Most neonatal HSV infection is the consequence of delivery of a neonate through an infected birth canal. The virus either invades the uterus following membrane rupture or contacts the fetus at delivery. Neonatal infection may be localized to the skin, eye, and mouth; involve the central nervous system or be disseminated.

Studies indicate a 30-50% risk of neonatal infection with a primary maternal infection near the time of delivery, but low risk (<1%) among women with recurrent maternal infection at term, or for those who acquire genital herpes during the first half of pregnancy. Neonatal mortality is 30% with disseminated disease and 4% with CNS disease. Approximately 20% of survivors of neonatal herpes have long-term neurologic sequelae.

## II. DIAGNOSIS

A clinical diagnosis of HSV infection may be confirmed by viral culture from skin lesions. However, there is a false-negative rate of 25% in primary infections. Culture of vulvar lesions has a low sensitivity, particularly in recurrent outbreaks or as lesions begin to heal.

- A. CDC guidelines recommend confirming the diagnosis of HSV with laboratory testing (culture or PCR or serology).
- B. CDC guidelines recommend determination of HSV serotype (HSV – 1 or HSV – 2) to aid in counseling.
- C. Clients with a new diagnosis of (or suspicion for) a STI should be offered concurrent STI screening.

## III. TREATMENT

- A. Clients with a positive test result or patients with symptoms and/or sexual contact with confirmed positive partner should be treated following the most recent CDC Sexually Transmitted Diseases Treatment Guidelines which can be accessed at CDC website:  
<http://www.cdc.gov/std/treatment/default.htm>
- B. Systemic antiviral drugs partially control the symptoms and signs of herpes episodes when used to treat first clinical episodes and recurrent episodes or when used as daily suppressive therapy. However, these drugs neither eradicate latent virus nor affect the risk, frequency, or severity of recurrences after the drug is discontinued.

## IV. SPECIAL TREATMENT CONSIDERATIONS

- A. The decision to use suppressive therapy depends on the frequency and severity of recurrent episodes. Therapy management must be individualized and should be assessed yearly. Prolonged therapy may be warranted.
- B. Once-daily valacyclovir suppressive therapy significantly reduces the risk of transmission of genital HSV among heterosexual HSV-2 discordant couples.
- C. When exposed to HIV, HSV-2 seropositive persons are at increased risk for HIV acquisition. Patients should be informed that suppressive antiviral therapy does not reduce the increased risk for HIV acquisition associated with HSV-2 infection.
- D. Acyclovir treatment late in pregnancy reduces the frequency of cesarean sections among women who have recurrent genital herpes by diminishing the frequency of recurrences at term.
- E. No data support the use of antiviral therapy among HSV seropositive women without a history of genital herpes.

## V. MANAGEMENT AND CLIENT COUNSELING/EDUCATION

- A. Advise the client to abstain from sexual contact during the prodromal period and
- B. while lesions are present.
- C. Advise the client to use latex condoms during asymptomatic periods to avoid
- D. transmission of the virus.

- E. Clients with HSV should inform their current partners that they have HSV and inform
- F. future partners before initiating a sexual relationship.
- G. Sex partners of HSV-infected persons should be advised that they might be infected even if they have no symptoms.
- H. Reproductive-aged clients should be counseled regarding the risks of neonatal herpes, including the recommendation to avoid exposure to HSV in pregnancy and possible need for chemoprophylaxis in pregnancy.

## VI. FOLLOW-UP

The client should be educated that if she becomes pregnant she should inform her obstetric health care provider of her past history of HSV.

## REFERENCES

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